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TYPE S9/MEDIUM, AB/1-17

9/AB/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

00011743978 BIOSIS NO.: 199900003638
Human psoriatic skin in organ culture: Comparison with normal skin exposed to exogenous growth factors and effects of an antibody to the EGF receptor
AUTHOR: Varani James (Reprint); Kang Sewon; Stoll Stefan; Elder James T
AUTHOR ADDRESS: Dep. Pathology, Univ. Mich., 1301 Catherine Road, Box 0602, Ann Arbor, MI 48109, USA**USA
JOURNAL: Pathobiology 66 (6): p253-259 Nov.-Dec., 1998 1998
MEDIUM: print
ISSN: 1015-2008
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Organ cultures were established from psoriatic lesional skin of 24 different individuals and maintained for 8 days under serum-free, growth-factor-free conditions. Nonlesional skin from 14 of the same individuals and normal skin from another 12 individuals were also maintained in organ culture. At the end of the incubation period, the tissues were fixed in formalin and examined histologically. Lesional skin continued to express features of psoriatic plaque, which included irregularly shaped epithelial cells arranged in a disorganized fashion, and elongation of the rete ridges with a thickening in their lower portion. Abnormal epidermal differentiation and separation of the upper epidermal layers from the lower layers was also a consistent feature. In contrast, nonlesional skin from psoriatic patients exhibited a histological appearance which resembled that of site-matched normal skin. When normal skin was exposed to a growth-factor-enriched culture medium during the 8-day incubation period, it exhibited a histological appearance similar to that of psoriatic skin. In addition to abnormal histological features, the psoriatic skin in organ culture released higher amounts of matrix metalloproteinase-9 (MMP-9; 92kD gelatinase B/type IV collagenase) into the culture fluid than either nonlesional skin or normal skin. Organ cultures of psoriatic lesional skin from 6 individuals were maintained for 8 days in the presence of an antibody to the human epidermal growth-factor (EGF) receptor. The abnormal histological features of the psoriatic tissue were partially ameliorated in the presence of the antibody. These data suggest that growth factors which act through the EGF receptor help to maintain the psoriatic phenotype in organ culture. They also suggest that organ culture may provide a useful tool with which to elucidate the pathophysiological mechanisms of altered keratinocyte proliferation and differentiation in psoriasis.

9/AB/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0009892592 BIOSIS NO.: 199598360425
Immunolocalization of epidermal growth factor and epidermal growth factor receptors in psoriatic epidermis
AUTHOR: Liu Baojun; Zhang; Haitao; Li; Shuqin
AUTHOR ADDRESS: Shenyang Miltary General Hosp., Shenyang 110015, China**

China
JOURNAL: Zhonghua Pifuke Zazhi 28 (2): p67-69 1995 1995
ISSN: 0412-4030
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: Chinese

ABSTRACT: We used an avidin-biotin-complex immunoperoxidase technique with anti-epidermal growth factor (EGF) polyclonal antibody and anti-EGF receptors (EGFR) monoclonal antibody to investigate the skin lesions from 20 patients with psoriasis vulgaris and normal skin from 10 healthy volunteers. Our results showed that: (1) The immunoreactive proteins of EGF and EGFR were primarily restricted to the basal cells and suprabasal keratinocytes in both normal epidermis and uninvolved psoriatic epidermis; (2) EGF and EGFR were distributed in all epidermal layers of active psoriatic lesions, and elevated levels of EGF and EGFR were seen in the mid and upper epidermal layers; (3) The immunoreactive proteins of EGF and EGFR were absent in the stratum corneum and decreased in the upper epidermal layers in regressing psoriatic lesions. It is suggested that EGF and EGFR may play an important role in the hyperproliferation and abnormal differentiation of keratinocytes seen in psoriasis.

9/AB/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0009351105 BIOSIS NO.: 199497372390
PUVA bath therapy strongly suppresses immunological and epidermal activation in psoriasis: A possible cellular basis for remittive therapy
AUTHOR: Vallat Val Pierre; Gilleaudeau Patricia; Battat Lisa; Wolfe Jonathan; Nabeya Reiko; Heftler Noah; Hodak Emmilia; Gottlieb Alice B (Reprint); Krueger James G
AUTHOR ADDRESS: Rockefeller Univ., 1230 York Ave., Box 178, New York, NY 10021-6399, USA**USA
JOURNAL: Journal of Experimental Medicine 180 (1): p283-296 1994 1994
ISSN: 0022-1007
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Psoriasis is characterized by alterations in both the epidermis and dermis of the skin. Epidermal keratinocytes display marked proliferative activation and differentiate along an "alternate" or "regenerative" pathway, while the dermis becomes infiltrated with leukocytes, particularly interleukin 2 (IL-2) receptor-bearing "activated" T cells. Psoralens, administered by the oral route, have therapeutic effects in psoriasis when photochemically activated by ultraviolet A light (PUVA therapy). Recently psoralen bath therapy has been introduced to more effectively deliver this agent to the diseased skin. We have correlated the efficacy of PUVA bath therapy with its effects on specific molecular and cellular parameters of disease, in 10 consecutive patients with recalcitrant psoriasis. Rapid clearing of lesions occurred in 8 out of 10 patients. Biopsies were taken from lesional and nonlesional skin before and after a single round of therapy, and observation was continued in our Clinical Research Center at The Rockefeller University. Enumeration of cycling keratinocytes with the Ki-67 monoclonal antibody showed that PUVA reduced cell proliferation by 73%. The pathological increase in insulin-like growth factor 1 (IGF-1) receptors was reversed, whereas epidermal growth factor (EGF) receptors,

which are also increased in psoriasis, remained unchanged. Keratinocyte proteins that are expressed in abnormal sites of the epidermis daring psoriasis, i.e., keratin 16, filaggrin, and involucrin, were, after PUVA treatment, localized to their normal sites. Epidermal and dermal T-lymphocytes (CD3+), as well as CD4+, CD8+, and IL-2 receptor+ subsets, were strongly suppressed by PUVA, with virtual elimination a IL-2 receptor+ T cells in some patients. Consistent with diminished lymphocyte activation, HLA-DR expression by epidermal keratinocytes was markedly reduced in treated skin. In comparison to cyclosporine treatment of psoriasis, PUVA therapy leads to more complete reversal of pathological epidermal and lymphocytic activation, changes which we propose to be the cellular basis for a more sustained remission of disease after PUVA treatment.

9/AB/4 (Item 4 from file: 5)

0009119715 BIOSIS NO.: 199497141000

Localization of annexins in normal and diseased human skin

AUTHOR: Bastian Boris C (Reprint); Van Der Piepen Ute; Roemisch Juergen; Paques Eric-P; Broecker Eva-Bettina
AUTHOR ADDRESS: Dep. Dermatol., Univ. Wurzburg, Med. Sch.,Josef-Schneider-Str. 2, D-97080 Wuerzburg, Germany**Germany

JOURNAL: Journal of Dermatological Science 6 (3): p225-234 1993 1993
ISSN: 0923-1811
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Annexins (AX) or lipocortins are a family of calcium and phospholipid binding proteins that have been implicated to play a role in the regulation of inflammation and cellular differentiation. To investigate a potential role of AX in skin disorders we studied the distribution of six different AX in normal human skin (NHS) and several inflammatory and hyperproliferative skin diseases. A distinct staining pattern could only be shown for AX-1 and AX-2. In NHS AX1-antibody (Ab) displayed a very strong reactivity with eccrine sweat ducts. In the diseases investigated we found a highly increased expression of AX-1 in keratinocytes (KCs) in the vincinity of inflammatory processes such as psoriasis. Furthermore, the AX-1 expression was increased in differentiated squamous cell carcinoma (SCC) whereas undifferentiated SCC and basal cell carcinoma were negative. AX-3, -4, -5, and -6 showed no distinctive expression pattern. Our data demonstrate an abnormal distribution of AX-1 in association with proliferating KCs under inflammatory and neoplastic conditions. Its pattern of reactivity shows similarities to the known distribution of the EGF-receptor kinase, which has been demonstrated to phosphorylate AX-1 with high activity in various cellular systems. These results support the concept that the appearance of AX-1 is linked to a certain level of KC differentiation.

9/AB/5 (Item 5 from file: 5)

0008760409 BIOSIS NO.: 199395062675

Induction of human microvascular endothelial tubular morphogenesis by human keratinocytes: Involvement of transforming growth factor-alpha

AUTHOR: Ono Mayumi (Reprint); Okamura Kazuki (Reprint); Nakayama Yoshifumi (Reprint); Tomita Mika; Sato Yasufumi (Reprint); Komatsu Yasuhiro; Kuwano Michihiko (Reprint)
AUTHOR ADDRESS: Dep. Biochem., Oita Med. Univ., Hasama-machi, Oita 879-55, Japan**Japan
JOURNAL: Biochemical and Biophysical Research Communications 189 (2): p 601-609 1992
ISSN: 0006-291X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Transforming growth factor-alpha(TGF-alpha), homologous to epidermal growth factor(EGF), is closely involved in hyperproliferation of human keratinocytes. Psoriasis is a common hyperproliferative skin disease characterized by hyperproliferation of keratinocytes and abnormal development of dermal capillary networks. In this study, we have examined whether keratinocytes could enhance angiogenesis. TGF-alpha or EGF efficiently stimulated formation of tubular-like structures of human omental microvascular endothelial (HOME) cells in type I collagen gels. Human keratinocytes produce TGF-alpha. To examine whether co-cultured keratinocytes could induce tubulogenesis of HOME cells in collagen gel, we have developed a co-culture system with human keratinocytes. Surprisingly, there appeared new development of many tubular-like structures of HOME cells in collagen gels when co-cultured with keratinocytes. This keratinocytes-dependent tubulogenesis was almost completely blocked when anti-TGF-alpha-antibody was present. The TGF-alpha molecules derived from keratinocytes appeared to enhance tubulogenesis of human microvascular endothelial cells. We propose the hypothesis that secretory TGF-alpha from human keratinocytes may promote an autocrine loop to proliferate the skin keratinocytes and also a paracrine loop to induce the skin angiogenesis.

9/AB/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0008419006 BIOSIS NO.: 199294120847
MOLECULAR CLONING AND EXPRESSION OF A NOVEL KERATINOCYTE PROTEIN PSORIASIS-ASSOCIATED FATTY ACID-BINDING PROTEIN PA-FABP THAT IS HIGHLY UP-REGULATED IN PSORIATIC SKIN AND THAT SHARES SIMILARITY TO FATTY ACID-BINDING PROTEINS
AUTHOR: MADSEN P (Reprint); RASMUSSEN H H; LEFFERS H; HONORE B; CELIS J E
AUTHOR ADDRESS: INST MED BIOCHEM, DANISH CENT HUMAN GENOME RES, OLE WORMS ALLE, BUILDING 170, UNIVERSITY PARK, DK-8000 AARHUS C, DENMARK**DENMARK
JOURNAL: Journal of Investigative Dermatology 99 (3): p299-305 1992
ISSN: 0022-202X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: ENGLISH

ABSTRACT: Analysis by means of two-dimensional (2D) gel electrophoresis of the protein patterns of normal and psoriatic unfractionated non-cultured keratinocytes has revealed a few low-molecular-weight proteins that are highly up-regulated in psoriatic skin. These include psoriasin; calgranulin B, also known as MRP 14, L1, or calprotectin; calgranulin A or MRP 8; and cystatin A or stefin A. Here, we have cloned and sequenced the cDNA (clone 1592) encoding a new member of this group of low-molecular-weight proteins [isoelectric focusing (IEF) SSP 3007 in the

keratinocyte 2D gel protein database] that we have termed PA-FABP (psoriasis-associated fatty acid-binding protein). The deduced sequence predicted a protein with molecular weight of 15,164 daltons and a calculated pI of 6.96, values that are close to those recorded in the keratinocyte 2D gel protein database. The protein comigrated with PA-FABP as determined by 2D gel analysis of [35S]-methionine-labeled proteins expressed by transformed human amnion (AMA) cells transfected with clone 1592 using the vaccinia virus expression system and reacted with a rabbit polyclonal antibody raised against 2D gel purified PA-FABP. Structural analysis of the amino acid sequence revealed 48%, 52%, and 56% identity to known low-molecular-weight fatty acid-binding proteins belonging to the FABP family. Northern blot analysis showed that PA-FABP mRNA is indeed highly up-regulated in psoriatic keratinocytes. The transcript is present in human cell lines of epithelial and lymphoid (Molt 4) origin but cannot be detected in normal or SV40 transformed MRC-5 fibroblasts. 2D gel protein analysis of normal primary keratinocytes cultured for at least 8 d under conditions that promoted incomplete terminal differentiation [serum-free keratinocyte (SKF) medium supplemented with epidermal growth factor (EGF), pituitary extract, and 10% fetal calf serum] revealed a strong up-regulation of PA-FABP, psoriasin, calgranulins A and B, and a few other proteins that are highly expressed in psoriatic skin. The levels of these proteins exceeded by far those observed in non-cultured normal keratinocytes implying that the culttured cells have followed an altered pattern of differentiation that resembles - at least in part - that of non-cultured psoriatic keratinocytes. The implications of these results for the study of psoriasis are discussed.

9/AB/7 (Item 7 from file: 5)

0008301674 BIOSIS NO.: 199294003515

THE INSULIN-LIKE GROWTH FACTOR I RECEPTOR IS OVEREXPRESSED IN PSORIATIC EPIDERMIS BUT IS DIFFERENTIALLY REGULATED FROM THE EPIDERMAL GROWTH FACTOR RECEPTOR

AUTHOR: KRANE J F (Reprint); GOTTLIEB A B; CARTER D M; KRUEGER J G
AUTHOR ADDRESS: LAB INVEST DERMATOL, ROCKEFELLER UNIV, 1230 YORK AVE, NEW YORK, NY 10021, USA**USA

JOURNAL: Journal of Experimental Medicine 175 (4): p1081-1090 1992
ISSN: 0022-1007
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: ENGLISH

ABSTRACT: Insulin-like growth factor I (IGF-I)/somatomedin C is an important mediator of keratinocyte growth in vitro, and the expression of IGF-I receptors in the basal layer of normal epidermis suggests that this growth pathway may function in the regulation of keratinocyte growth in vivo as well. The pattern of IGF-I receptor expression in normal skin is distinct from that of the epidermal growth factor (EGF) receptor, suggesting that these receptors might be differentially regulated. The purpose of this study was to obtain a better understanding of IGF-I receptor function in the skin by examining IGF-I receptor expression in psoriatic epidermis and in cultured human keratinocytes. Our findings indicate that IGF-I receptor expression is increased in psoriasis as measured by protein tyrosine kinase assays of biopsy extracts and by immunohistochemical staining with an IGF-I receptor-specific monoclonal antibody. Unlike EGF receptor expression, which is also increased in psoriatic epidermis, the pattern of IGF-I receptor expression corresponds

closely with the increased size of the keratinocyte proliferative compartment in psoriasis. Biochemical agents that diminish EGF receptor ligand binding (phorbol ester or calcium ionophore treatment) produce opposite effects on the IGF-I receptor. These results suggest that cellular expression and differential regulation of both growth factor receptor systems may control critical aspects of epidermal proliferation or function.

9/AB/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0006590332 BIOSIS NO.: 198987038223
DISTRIBUTION OF EGF RECEPTOR EXPRESSING AND DNA REPLICATING EPIDERMAL CELLS IN PSORIASIS VULGARIS AND BOWEN'S DISEASE
AUTHOR: AMAGAI M (Reprint); OZAWA S; UEDA M; NISHIKAWA T; ABE O; SHIMIZU N
AUTHOR ADDRESS: DEP MOL BIOL, KEIO UNIV SCH MED, 35 SHINANOMACHI, SHINJUKU-KU, TOKYO 160, JPN**JAPAN
JOURNAL: British Journal of Dermatology 119 (5): p661-668 1988
ISSN: 0007-0963
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: ENGLISH

ABSTRACT: We have examined the localization of DNA replicating cells and EGE receptor-expressing cells in the epidermis of psoriasis vulgaris, a benign hyperproliferative skin disease, and Bowen's disease, a pre-malignant hyperproliferative skin disease, and normal skin. DNA replicating cells were detected by anti-BrdU monoclonal antibody after incubating tissue sections with BrdU, and EGF receptors were detected by the anti-EGF receptor monoclonal antibody B4G7. In normal skin, DNA replicating cells were localized exclusively in the basal and suprabasal layers. EGF receptor expression was observed most strongly in the basal and parabasal layers, but diminished gradually towards the upper squamous layer. In psoriatic skin, DNA replicating cells were also localized in the basal and parabasal layers, but the number of these mitotic cells was about 10 times higher than in normal skin. In this case, more EGF receptors were detected in all viable layers of the epidermis. Apparently normal skin adjacent to psoriasis lesions showed persistent expression of EGF receptors in the upper squamous later without an increased number of DNA replicating cells in the basal and parabasal layers. In Bowen's disease, DNA replicating cells and EGF receptor expressing cells were distributed in all layers of the epidermis. These findings indicate that the increased production of EGF receptors may be, in part, responsible for the hyperproliferative state of the epidermis and that cells in the upper squamous layer of psoriasis may have lost a mechanism by which EGF receptor expression is diminished thus allowing differentiation. This altered process of EGF receptor production may be involved in the onset of psoriasis vulgaris.

9/AB/9 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

12713145 EMBASE No: 2004310856
In this issue
Elder J.T.
J.T. Elder, University of Michigan, Medical Center, Ann Arbor Veterans

Affairs Hospital, Ann Arbor, MI United States
Journal of Investigative Dermatology (J. INVEST. DERMATOL.) (United States) 2004, 123/2 (vi-vii)
CODEN: JIDEA ISSN: 0022-202X
DOCUMENT TYPE: Journal ; Editorial
LANGUAGE: ENGLISH

9/AB/10 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

06467279 EMBASE No: 1996132953
Overexpression of amphiregulin, a major autocrine growth factor for cultured human keratinocytes, in hyperproliferative skin diseases
Piepkorn M.
Dermatology Division, University of Washington, Box 356524 Health Sciences,Seattle, WA 98195 United States
American Journal of Dermatopathology (AM. J. DERMATOPATHOL.) (United States) 1996, 18/2 (165-171)
CODEN: AJDOD ISSN: 0193-1091
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

Previous studies have indicated that amphiregulin is a major autocrine growth factor for cultured human keratinocytes. Its overexpression could therefore be important in hyperproliferative skin diseases. The purpose of this preliminary study was to determine if there is upregulation of amphiregulin protein in those disorders. A variety of lesions was surveyed for qualitative alterations in its immunostaining with an anti-amphiregulin monoclonal antibody. Amphiregulin was barely detectable in the epidermis of normal controls, although there was random nuclear staining of keratinocytes, and the epidermal appendages, especially sebaceous glands, were usually reactive. In contrast, psoriatic lesions exhibited prominent cytoplasmic staining of basal and spinous keratinocytes. Somewhat increased reactivity was also evident in actinic keratoses, in nests of squamous carcinoma cells, and in verrucae. Adnexal tumors were often strongly stained. Whereas basal cell carcinomas were nonreactive, staining was present in adjacent epidermis. Similarly, the melanocytes of nevi and melanoma were nonreactive but there was increased staining in contiguous keratinocytes. The pattern of amphiregulin immunostaining suggests a role for the protein in the aberrant keratinocyte growth of hyperproliferative disorders.

9/AB/11 (Item 3 from file: 73)
DIALOG(R)File 73:EMBASE

06033610 EMBASE No: 1995063848
Cd44 isoforms containing exon V3 are responsible for the presentation of heparin-binding growth factor
Bennett K.L.; Jackson D.G.; Simon J.C.; Tanczos E.; Peach R.; Modrell B.; Stamenkovic I.; Plowman G.; Aruffo A.
B. M. Squibb Pharmaceut. Res. Inst., 3005 First Avenue,Seattle, WA 98121 United States
Journal of Cell Biology (J. CELL BIOL.) (United States) 1995, 128/4 (687-698)
CODEN: JCLBA ISSN: 0021-9525
DOCUMENT TYPE: Journal; Article

LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

Glycosaminoglycan-modified isoforms of CD44 have been implicated in growth factor presentation at sites of inflammation. In the present study we show that COS cell transfectants expressing CD44 isoforms containing the alternatively spliced exon V3 are modified with heparan sulfate (HS). Binding studies with three HS-binding growth factors, basic-fibroblast growth factor (b-FGF), heparin binding-epidermal growth factor (HB-EGF), and amphiregulin, showed that the HS-modified CD44 isoforms are able to bind to b-FGF and HB- EGF, but not AR. b-FGF and HB-EGF binding to HS-modified CD44 was eliminated by pretreating the protein with heparitinase or by blocking with free heparin. HS-modified CD44 immunoprecipitated from keratinocytes, which express a CD44 isoform containing V3, also bound to b-FGF. We examined whether HS-modified CD44 isoforms were expressed by activated endothelial cells where they might present HS-binding growth factors to leukocytes during an inflammatory response. PCR and antibody-binding studies showed that activated cultured endothelial cells only express the CD44H isoform which does not contain any of the variably spliced exons including V3. Immunohistological studies with antibodies directed to CD44 extracellular domains encoded by the variably spliced exons showed that vascular endothelial cells in inflamed skin tissue sections do not express CD44 spliced variants. Keratinocytes, monocytes, and dendritic cells in the same specimens were found to express variably spliced CD44. sup 3sup 5SOinf 4sup -sup 2-labeling experiments demonstrated that activated cultured endothelial cells do not express detectable levels of chondroitin sulfate or HS-modified CD44. Our results suggest that one of the functions of CD44 isoforms expressing V3 is to bind and present a subset of HS-binding proteins. Furthermore, it is probable that HS-modified CD44 is involved in the presentation of HS-binding proteins by keratinocytes in inflamed skin. However, our data suggests that CD44 is not likely to be the proteoglycan principally involved in presenting HS-binding growth factors to leukocytes on the vascular cell wall.

9/AB/12 (Item 1 from file: 155)

09602687 PMID: 1836217

Lipocortin I (annexin I) is preferentially localized on the plasma membrane in keratinocytes of psoriatic lesional epidermis as shown by immunofluorescence microscopy.

Kitajima Y; Owada M K; Mitsui H; Yaoita H
Department of Dermatology, Jichi Medical School, Tochigiken, Japan.

Journal of investigative dermatology (UNITED STATES) Dec 1991, 97 (6) p1032-8, ISSN 0022-202X Journal Code: 0426720
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

Lipocortin I (LPC-I, also called annexin I) is a 35-kD protein that binds phospholipids and actin in a Ca(++)-dependent manner. It is also a major substrate for EGF receptor/kinase and protein kinase C, and a putative inhibitor of phospholipase A2, which produces chemical mediators to cause inflammation. Psoriasis (PS) is an inflammatory skin disease characterized by a rapid turnover of keratinocytes and a defect in keratinization with increased activities of phospholipase C and A2, and EGF receptor. To understand the mechanism of the PS lesion formation and the function of

LPC-I, its distribution was studied in the epidermis of PS, subacute eczema and normal skin, and in tumor cells of seborrheic keratosis and Bowen's disease. This study involved immunofluorescence and immunoblotting using affinity-purified polyclonal and monoclonal antibodies specific to LPC-I and to its Ca(++)-bound form. In normal, nonlesional PS and subacute eczema epidermis, LPC-I was detected mainly in the cytoplasm of the suprabasal cells, although it was on the inner aspects of the plasma membrane in some parts of the granular layer. In lesional epidermis of PS, it was localized mainly on the inner aspects of the plasma membrane, but not in the cytoplasm of the whole suprabasal cells as the Ca(++)-bound form, indicating a preferential localization on the plasma membrane. This membrane-binding of LPC-I was also observed in seborrheic keratosis, but not in Bowen's disease. These results suggest that the binding of LPC-I to the plasma membrane occurs actually in living cells, plays a role, not necessarily disease specific, in the PS lesion formation, and has some relevance to normal or abnormal differentiation of keratinocytes.

9/AB/13 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08213374 PMID: 3279155
Detection of transforming growth factor alpha in normal, malignant, and hyperproliferative human keratinocytes.
Gottlieb A B; Chang C K; Posnett D N; Fanelli B; Tam J P
Laboratory of Immunology, Rockefeller University, New York.
Journal of experimental medicine (UNITED STATES) Feb 1 1988, 167 (2) p670-5, ISSN 0022-1007 Journal Code: 2985109R
Contract/Grant No.: AI-19080; AI; NIAID; AR-35676; AR; NIAMS; CA-42046; CA; NCI; +
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Transforming growth factor alpha (TGF-alpha) is a 50-amino acid peptide, previously demonstrated only in transformed cell lines and human tumors, which is structurally homologous to epidermal growth factor (EGF). TGF-alpha expression in keratinocytes from normal individuals, patients with psoriasis, and patients with malignant skin diseases was investigated using an mAb raised against synthetic human TGF-alpha. mAb A1.5 reacted with TGF-alpha, but not EGF, in a sensitive ELISA. Keratinocytes in eight nodular basal cell carcinomas, one morpheic basal cell carcinoma, and one squamous cell carcinoma demonstrated intense membranous immunoperoxidase staining with mAb A1.5. Of even greater interest was the observation that the overlying normal epidermis, as well as the epidermis from five normal skin specimens, were stained by the mAb. Keratinocytes in plaques from 18 psoriasis patients were more intensely stained than those from normal skin. Cultured normal keratinocytes demonstrated membranous staining with mAb A1.5. Absorption of mAb A1.5 with synthetic human TGF-alpha completely removed the reactivity of mAb A1.5 with both basal cell tumors and normal epidermis. The demonstration of TGF-alpha in normal keratinocytes suggests that it plays a role in normal keratinocyte growth, wound healing, and in the pathogenesis of acanthosis.

9/AB/14 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

141189645 CA: 141(12)189645h PATENT
Humanized or chimeric anti-amphiregulin antibodies, fragments and immunoconjugates for treat cancer or proliferative disease and psoriasis
INVENTOR(AUTHOR): Landolfi, Nicholas F.; Tsurushita, Naoya; Hinton, Paul R.; Kumar, Shankar
LOCATION: USA
ASSIGNEE: Protein Design Labs Inc.
PATENT: PCT International ; WO 200468931 A2 DATE: 20040819
APPLICATION: WO 2004US4176 (20040206) *US PV445640 (20030207) *US PV533901 (20031230)
PAGES: 99 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-031/00A
DESIGNATED COUNTRIES: AE; AE; AG; AL; AL; AM; AM; AM; AT; AT; AU; AZ; AZ; BA; BB; BG; BG; BR; BR; BW; BY; BY; BZ; BZ; CA; CH; CN; CN; CO; CO; CR; CR; CU; CU; CZ; CZ; DE; DE; DK; DK; DM; DZ; EC; EC; EE; EE; EG; ES; ES; FI; FI; GB; GD; GE; GE; GH; GM; HR; HR; HU; HU; ID; IL; IN; IS; JP; JP; KE; KE; KG; KG; KP; KP; KP; KR; KR; KZ; KZ; KZ; LC; LK; LR; LS; LS; LT; LU; LV; MA; MD; MD; MG; MK; MN; MW; MX; MX; MZ; MZ; NA; NI DESIGNATED REGIONAL: BW; GH; GM ; KE; LS; MW; MZ; SD; SL; SZ; TZ; UG; ZM; ZW; AT; BE; BG; CH; CY; CZ; DE; DK; EE; ES; FI; FR; GB; GR; HU; IE; IT; LU; MC; NL; PT; RO; SE; SI; SK; TR; BF; BJ; CF; CG; CI; CM; GA; GN; GQ; GW; ML; MR; NE; SN; TD; TG; BF; BJ; CF; CG; CI; CM; GA; GN; GQ; GW; ML; MR; NE; SN; TD; TG

9/AB/15 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

139212354 CA: 139(14)212354g PATENT
Methods and compositions for the therapeutic use of contact inhibitory factor (CIF)
INVENTOR(AUTHOR): Lipkin, George; Rosenberg, Martin Jay
LOCATION: USA
ASSIGNEE: New York University
PATENT: PCT International ; WO 200372737 A2 DATE: 20030904
APPLICATION: WO 2003US5563 (20030224) *US PV359053 (20020222) *US PV386570 (20020605)
PAGES: 26 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: C12N-000/A
DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; BZ; CA; CH; CN; CO; CR; CU; CZ; DE; DK; DM; DZ; EC; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; MZ; NO; NZ; OM; PH; PL; PT; RO; RU; SC; SD; SE; SG; SK; SL; TJ; TM; TN; TR; TT; TZ; UA; UG; US; UZ; VC; VN; YU; ZA; ZM; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; GM; KE; LS ; MW; MZ; SD; SL; SZ; TZ; UG; ZM; ZW; AT; BE; BG; CH; CY; CZ; DE; DK; EE; ES; FI; FR; GB; GR; HU; IE; IT; LU; MC; NL; PT; SE; SI; SK; TR; BF; BJ; CF; CG; CI; CM; GA; GN; GQ; GW; ML; MR; NE; SN; TD; TG

9/AB/16 (Item 3 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

136161345 CA: 136(11)161345h PATENT
Treatment of hyperproliferative diseases with epidermal growth factor receptor antagonists
INVENTOR(AUTHOR): Teufel, Thomas
LOCATION: USA
ASSIGNEE: Imclone Systems Incorporated
PATENT: PCT International ; WO 200211677 A2 DATE: 20020214

APPLICATION: WO 2001US41647 (20010809) *US 635974 (20000809)
PAGES: 28 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-000/A
DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; BZ; CA; CH; CN; CO; CR; CU; CZ; DE; DK; DM; DZ; EC; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; MZ; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; TZ; UA; UG; US; UZ; VN; YU; ZA; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; GM; KE; LS; MW; MZ; SD; SL; SZ ; TZ; UG; ZW; AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; TR; BF; BJ; CF; CG; CI; CM; GA; GN; GQ; GW; ML; MR; NE; SN; TD; TG

9/AB/17 (Item 4 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

124340904 CA: 124(25)340904p PATENT
Methods and bifunctional ligands for specific tumor inhibition by blood coagulation in tumor vasculature
INVENTOR(AUTHOR): Thorpe, Philip E.; Edgington, Thomas S.
LOCATION: USA
ASSIGNEE: Univ. of Texas System; Scripps Res. Inst.
PATENT: PCT International ; WO 9601653 A1 DATE: 960125
APPLICATION: WO 95US7439 (950607) *US 273567 (940711)
PAGES: 325 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-047/48A
DESIGNATED COUNTRIES: AM; AT; AU; BB; BG; BR; BY; CA; CH; CN; CZ; DE; DK; EE; ES; FI; GB; GE; HU; IS; JP; KE; KG; KP; KR; KZ; LK; LR; LT; LU; LV; MD; MG; MN; MW; MX; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; TJ; TT; UA
DESIGNATED REGIONAL: KE; MW; SD; SZ; UG; AT; BE; CH; DE; DK; ES; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN; ML; MR; NE; SN; TD; TG
?

Set	Items	Description
S1	68289	EGF
S2	68894	S1 OR AMPHIREGULIN
S3	3056	S2 AND HEPARIN
S4	371	S3 AND ANTIBODY
S5	2	S4 AND PSORIASIS
S6	68972	S2 OR HEPARIN BINDING
S7	6249	S6 AND ANTIBODY
S8	34	S7 AND PSORIASIS
S9	17	RD S8 (unique items)

?

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Date: 2/24/2005

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Inventor Name Search Result

Your Search was:

Last Name = LANDOLFI
First Name = NICHOLAS

Application#	Patent#	Status	Date Filed	Title	Inventor Name
09718993	Not Issued	161	11/22/2000	HUMANIZED IMMUNOGLOBULINS	LANDOLFI, NICHOLAS F.
09718998	Not Issued	041	11/22/2000	HUMANIZED IMMUNOGLOBULINS	LANDOLFI, NICHOLAS F.
09992524	Not Issued	071	11/13/2001	HUMANIZED ANTIBODIES TO GAMMA-INTERFERON	LANDOLFI, NICHOLAS F.
10389155	Not Issued	030	03/13/2003	HUMANIZED IMMUNOGLOBULINS	LANDOLFI, NICHOLAS F.
10389417	Not Issued	030	03/13/2003	HUMANIZED IMMUNOGLOBULINS	LANDOLFI, NICHOLAS F.
10394458	Not Issued	168	03/20/2003	HUMANIZED IMMUNOGLOBULINS	LANDOLFI, NICHOLAS F.
10452357	Not Issued	030	05/30/2003	HUMANIZED IMMUNOGLOBULINS	LANDOLFI, NICHOLAS F.
10774076	Not Issued	071	02/06/2004	AMPHIREGULIN ANTIBODIES AND THEIR USE TO TREAT CANCER AND PSORIASIS	LANDOLFI, NICHOLAS F.
10842011	Not Issued	030	05/07/2004	THERAPEUTIC USE OF ANTI-CS1 ANTIBODIES	LANDOLFI, NICHOLAS F.
07532267	Not Issued	166	06/01/1990	CHIMERIC LIGAND/IMMUNOGLOBULIN MOLECULES AND THEIR USES	LANDOLFI, NICHOLAS F.
07728962	Not Issued	161	07/12/1991	PRODUCTION OF HUMAN ANTIBODIES	LANDOLFI, NICHOLAS F.
07983949	Not Issued	166	12/01/1992	HUMANIZED ANTIBODIES REACTIVE WITH CD18	LANDOLFI, NICHOLAS F.
08076263	5349053	250	06/10/1993	CHIMERIC LIGAND/IMMUNOGLOBULIN MOLECULES AND THEIR USES	LANDOLFI, NICHOLAS F.
08304646	Not Issued	161	09/12/1994	HUMANIZED ANTIBODIES REACTIVE WITH CD18	LANDOLFI, NICHOLAS F.
08477728	5585089	150	06/07/1995	HUMANIZED	LANDOLFI,

				IMMUNOGLOBULINS	NICHOLAS F.
08484537	6180370	150	06/07/1995	HUMANIZED IMMINOGLOBULINS AND METHODS OF MAKING THE SAME	LANDOLFI, NICHOLAS F.
08487200	5693762	150	06/07/1995	HUMANIZED IMMUNOGLOBULINS	LANDOLFI, NICHOLAS F.
08621751	5882644	150	03/22/1996	MONOCLONAL ANTIBODIES SPECIFIC FOR THE PLATELET DERIVED GROWTH FACTOR BETA RECEPTOR AND METHODS OF USE THEREOF	LANDOLFI, NICHOLAS F.
09325000	Not Issued	168	06/01/1999	HUMANIZED IMMUNOGLOBULINS	LANDOLFI, NICHOLAS F.
09450520	6329511	150	11/29/1999	HUMANIZED ANTIBODIES TO GAMMA-INTERFERON	LANDOLFI, NICHOLAS F.
60110523	Not Issued	159	12/01/1998	HUMANIZED ANTIBODIES TO GAMMA-INTERFERON	LANDOLFI, NICHOLAS F.

Inventor Search Completed: No Records to Display.

Search Another: Inventor **Last Name** landolfi **First Name** nicholas Search

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Inventor Name Search Result

Your Search was:

Last Name = TSURUSHITA
First Name = NAOYA

Application#	Patent#	Status	Date Filed	Title	Inventor Name
07988255	Not Issued	161	12/09/1992	CONSTRUCTION OF HUMAN IMMUNOGLOBULIN COMBINATORIAL LIBRARY BASED ON ARTIFICIAL V GENES COMPRISING GENOMIC V SEGMENTS AND SYNTHETIC CDR3 FRAGMENTS	TSURUSHITA, NAOYA
09450520	6329511	150	11/29/1999	HUMANIZED ANTIBODIES TO GAMMA-INTERFERON	TSURUSHITA, NAOYA
09772103	Not Issued	071	01/26/2001	ANTIBODIES AGAINST CTLA4 AND USES THEREFOR	TSURUSHITA, NAOYA
09992524	Not Issued	071	11/13/2001	HUMANIZED ANTIBODIES TO GAMMA-INTERFERON	TSURUSHITA, NAOYA
10226435	Not Issued	080	08/22/2002	HUMANIZED ANTIBODIES THAT SEQUESTER ABETA PEPTIDE	TSURUSHITA, NAOYA
10371483	Not Issued	030	02/21/2003	ANTI-CCR5 ANTIBODY	TSURUSHITA, NAOYA
10476265	Not Issued	030	10/22/2003	HUMANIZED ANTIBODIES	TSURUSHITA, NAOYA
10484280	Not Issued	030	01/15/2004	INTERLEUKIN-1 BETA ANTIBODIES	TSURUSHITA, NAOYA
10486908	Not Issued	030	02/17/2004	ASSAY METHOD FOR ALZHEIMER'S DISEASE	TSURUSHITA, NAOYA
10487322	Not Issued	030	02/17/2004	ANTI-ABETA ANTIBODIES	TSURUSHITA, NAOYA
10687118	Not Issued	030	10/15/2003	ALTERATION OF FCRN BINDING AFFINITIES OR SERUM HALF-LIVES OF ANTIBODIES BY MUTAGENESIS	TSURUSHITA, NAOYA

10774076	Not Issued	071	02/06/2004	AMPHIREGULIN ANTIBODIES AND THEIR USE TO TREAT CANCER AND PSORIASIS	TSURUSHITA, NAOYA
10788625	Not Issued	030	02/26/2004	HUMANIZED CHICKEN ANTIBODIES	TSURUSHITA, NAOYA
10822300	Not Issued	030	04/09/2004	ALTERATION OF FCRN BINDING AFFINITIES OR SERUM HALF-LIVES OF ANTIBODIES BY MUTAGENESIS	TSURUSHITA, NAOYA
10966673	Not Issued	019	10/15/2004	ALTERATION OF FC-FUSION PROTEIN SERUM HALF-LIVES BY MUTAGENESIS	TSURUSHITA, NAOYA
60110523	Not Issued	159	12/01/1998	HUMANIZED ANTIBODIES TO GAMMA-INTERFERON	TSURUSHITA, NAOYA
60120312	Not Issued	159	02/16/1999	NOVEL CHEMOKINE AND USES THEREOF	TSURUSHITA, NAOYA
60178473	Not Issued	159	01/27/2000	ANTIBODIES AGAINST CTLA4 AND USES THEREFOR	TSURUSHITA, NAOYA
60287539	Not Issued	159	04/30/2001	HUMANIZED ANTIBODIES	TSURUSHITA, NAOYA
60307973	Not Issued	159	07/26/2001	INTERLEUKIN-1 BETA ANTIBODIES	TSURUSHITA, NAOYA
60312278	Not Issued	159	08/14/2001	INTERLEUKIN-1 BETA ANTIBODIES	TSURUSHITA, NAOYA
60313224	Not Issued	159	08/17/2001	ANTI-AB ANTIBODIES	TSURUSHITA, NAOYA
60418972	Not Issued	159	10/15/2002	ALTERATION OF FCRN BINDING AFFINITIES OR SERUM HALF-LIVES OF ANTIBODIES BY MUTAGENESIS	TSURUSHITA, NAOYA
60451622	Not Issued	159	02/28/2003	HUMANIZED CHICKEN ANTIBODIES	TSURUSHITA, NAOYA
60462014	Not Issued	159	04/10/2003	ALTERATION OF FCRN BINDING AFFINITIES OR SERUM HALF-LIVES OF ANTIBODIES BY MUTAGENESIS	TSURUSHITA, NAOYA
60475762	Not Issued	159	06/03/2003	ALTERATION OF FCRN BINDING AFFINITIES OR SERUM HALF-LIVES OF ANTIBODIES BY	TSURUSHITA, NAOYA

				MUTAGENESIS	
60489187	Not Issued	159	07/21/2003	EXPRESSION OF PERIOSTIN IS INDUCED BY IL-4 AND IL-13 IN PRIMARY ENDOTHELIAL CELLS	TSURUSHITA, NAOYA
60497474	Not Issued	159	08/21/2003	HUMANIZED ANTI-IP-10 ANTIBODIES AND METHODS OF USING THEREOF FOR THE TREATMENT OF INFLAMMATORY BOWEL DISEASES	TSURUSHITA, NAOYA
60499048	Not Issued	159	08/29/2003	ALTERATION OF SERUM HALF-LIVES OF ANTIBODIES BY MUTAGENESIS	TSURUSHITA, NAOYA
60511687	Not Issued	159	10/15/2003	ALTERATION OF FC-FUSION PROTEIN SERUM HALF-LIVES BY MUTAGENESIS	TSURUSHITA, NAOYA
60511688	Not Issued	159	10/15/2003	ALTERATION OF ANTIBODY COMPLEMENT DEPENDENT CYTOXICITY (CDC) ACTIVITY BY MUTAGENESIS	TSURUSHITA, NAOYA
60527882	Not Issued	159	12/04/2003	HUMANIZED ANTI-IP-10 ANTIBODIES AND METHODS OF USING THEREOF FOR THE TREATMENT OF INFLAMMATORY BOWEL DISEASES	TSURUSHITA, NAOYA
60562627	Not Issued	020	04/14/2004	ALTERATION OF FC-FUSION PROTEIN SERUM HALF-LIVES BY MUTAGENESIS	TSURUSHITA, NAOYA

Inventor Search Completed: No Records to Display.

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Inventor Name Search Result

Your Search was:

Last Name = HINTON
First Name = PAUL

Application#	Patent#	Status	Date Filed	Title	Inventor Name
10450384	Not Issued	030	12/24/2003	SILENCED ANTI-CD28 ANTIBODIES AND USE THEREOF	HINTON, PAUL
60178473	Not Issued	159	01/27/2000	ANTIBODIES AGAINST CTLA4 AND USES THEREFOR	HINTON, PAUL
60255155	Not Issued	159	12/14/2000	SILENCED ANTI-CD28 ANTIBODIES AND USE THEREOF	HINTON, PAUL
60287653	Not Issued	159	04/30/2001	HUMANIZED ANTIBODIES	HINTON, PAUL
60418972	Not Issued	159	10/15/2002	ALTERATION OF FCRN BINDING AFFINITIES OR SERUM HALF-LIVES OF ANTIBODIES BY MUTAGENESIS	HINTON, PAUL
08458666	6218149	150	06/02/1995	ANTIBODIES HAVING MODIFIED CARBOHYDRATE CONTENT AND METHODS OF PREPARATION AND USE	HINTON, PAUL R
09772103	Not Issued	071	01/26/2001	ANTIBODIES AGAINST CTLA4 AND USES THEREFOR	HINTON, PAUL R.
09835461	Not Issued	161	04/16/2001	ANTIBODIES HAVING MODIFIED CARBOHYDRATE CONTENT AND METHODS OF PREPARATION AND USE	HINTON, PAUL R.
10371483	Not Issued	030	02/21/2003	ANTI-CCR5 ANTIBODY	HINTON, PAUL R.
10687118	Not Issued	030	10/15/2003	ALTERATION OF FCRN BINDING AFFINITIES OR SERUM HALF-LIVES OF ANTIBODIES BY MUTAGENESIS	HINTON, PAUL R.
10774076	Not	071	02/06/2004	AMPHIREGULIN ANTIBODIES	HINTON, PAUL R.

	Issued			AND THEIR USE TO TREAT CANCER AND PSORIASIS	
10822300	Not Issued	030	04/09/2004	ALTERATION OF FCRN BINDING AFFINITIES OR SERUM HALF-LIVES OF ANTIBODIES BY MUTAGENESIS	HINTON, PAUL R.
10966673	Not Issued	019	10/15/2004	ALTERATION OF FC-FUSION PROTEIN SERUM HALF-LIVES BY MUTAGENESIS	HINTON, PAUL R.
60462014	Not Issued	159	04/10/2003	ALTERATION OF FCRN BINDING AFFINITIES OR SERUM HALF-LIVES OF ANTIBODIES BY MUTAGENESIS	HINTON, PAUL R.
60475762	Not Issued	159	06/03/2003	ALTERATION OF FCRN BINDING AFFINITIES OR SERUM HALF-LIVES OF ANTIBODIES BY MUTAGENESIS	HINTON, PAUL R.
60499048	Not Issued	159	08/29/2003	ALTERATION OF SERUM HALF-LIVES OF ANTIBODIES BY MUTAGENESIS	HINTON, PAUL R.
60511687	Not Issued	159	10/15/2003	ALTERATION OF FC-FUSION PROTEIN SERUM HALF-LIVES BY MUTAGENESIS	HINTON, PAUL R.
60511688	Not Issued	159	10/15/2003	ALTERATION OF ANTIBODY COMPLEMENT DEPENDENT CYTOXICITY (CDC) ACTIVITY BY MUTAGENESIS	HINTON, PAUL R.
60562627	Not Issued	020	04/14/2004	ALTERATION OF FC-FUSION PROTEIN SERUM HALF-LIVES BY MUTAGENESIS	HINTON, PAUL R.
07244744	Not Issued	166	09/15/1988	ANTIBODIES HAVING MODIFIED CARBOHYDRATE CONTENT AND METHODS OF PREPARATION AND USE	HINTON, PAUL R.
07260558	Not Issued	166	10/17/1988	ANTI-LEU 3A AMINO ACID SEQUENCE	HINTON, PAUL R.
07938557	Not Issued	166	08/28/1992	ANTIBODIES HAVING MODIFIED CARBOHYDRATE CONTENT AND METHODS OF PREPARATION AND USE	HINTON, PAUL R.
08251529	Not Issued	166	05/31/1994	ANTIBODIES HAVING MODIFIED CARBOHYDRATE	HINTON, PAUL R.

				CONTENT AND METHODS OF PREPARATION AND USE	
10497475	Not Issued	019	01/01/0001	HUMANIZED ANTIBODIES	HINTON, PAUL ROBERT

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10425195	Not Issued	030	04/29/2003	METHOD AND SYSTEM FOR GENERATING DOCUMENT PACKAGES FOR COMPLEX ENGINEERED EQUIPMENT AND MIXED APPARATUS ORDERS	KUMAR, SHANKAR
10774076	Not Issued	071	02/06/2004	AMPHIREGULIN ANTIBODIES AND THEIR USE TO TREAT CANCER AND PSORIASIS	KUMAR, SHANKAR
10788625	Not Issued	030	02/26/2004	HUMANIZED CHICKEN ANTIBODIES	KUMAR, SHANKAR

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